

# **EFFECTS OF AMNIOINFUSION IN MECONIUM STAINED AMNIOTIC FLUID**

*Dissertation submitted for*

**Doctor of Medicine**

**(Obstetrics and Gynecology)**

**Government Kilpauk Medical College, Chennai 600 010.**



**The Tamilnadu Dr. M G R University,**

**Chennai, Tamilnadu**

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## **DECLARATION**

I, hereby declare that this dissertation entitled 'Effects of Amnioinfusion in Meconium stained amniotic fluid' has been prepared by me under the direct guidance and supervision of Dr. Indrani Padmanabhan, Professor, Department of Obstetrics and Gynecology, Government Kilpauk Medical College, Chennai, in partial fulfillment of regulations of The Tamilnadu Dr. M G R Medical University, Chennai, for the award of Doctor of Medicine in Obstetrics and Gynecology.

This has not been submitted previously by me to this university or any other university for the award of any degree or diploma.

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This work has not formed the basis for award of any other degree or diploma to the candidate.

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## 1. INTRODUCTION

Amniotic fluid serves several functions, such as providing a medium for fetal growth and development, protecting the fetus from external trauma and maintaining intrauterine temperature.

Meconium stained amniotic fluid occurs in 10% of all pregnancies: in 5% of these (i.e. 1 : 200 of all pregnancies) the meconium is aspirated in to the lungs of the fetus. Meconium aspiration syndrome contributes to neonatal death in approximately 10% of babies who aspirate (i.e. 1 : 2000 of all pregnancies). In the past, it was considered as a sign of fetal distress occurring only in response to hypoxia. But in most cases meconium in the amniotic fluid is benign and associated with term pregnancies. But there is a general agreement that the presence of meconium stained amniotic fluid is associated with increase in perinatal mortality and morbidity. Many investigators does not believe that presence of meconium predict a poor out come unless it is accompanied by other signs of fetal distress.

How ever if aspirated by the fetus before or during delivery, meconium can obstruct the airways, interfere with gas transfer and cause respiratory difficulties.

The increased perinatal mortality and morbidity in the presence of meconium stained amniotic fluid is mainly due to meconium aspiration syndrome occurring in 2 – 10% of these neonates. 50 – 60% of infants whose amniotic fluid was meconium stained was reported to have meconium in their tracheas.



Treatment strategies of deep suctioning of hypopharynx and nares during delivery and immediate tracheal suctioning after birth have been successful in reducing meconium aspiration syndrome. But despite aggressive airway cleaning, meconium aspiration is not always prevented, the causative factor being inutero aspiration.

Amnioinfusion is a simple technique, which dilutes the amniotic fluid and has been studied as an additional tool to prevent meconium aspiration. It helps by physical dilution of the thick meconium and hence decreases the toxic effects of aspiration of thick meconium. It also increases the fluid around the fetus and thereby decreases cord compression and hence fetal hypoxia when present.

## **2. AIM OF THE STUDY**

To evaluate the effect of amnioinfusion in meconium stained amniotic fluid on

- the incidence of intrapartum fetal heart rate abnormalities
- the nature of labour and incidence of instrumental deliveries
- the neonatal outcome
- the incidence of Meconium aspiration syndrome

### **3. REVIEW OF LITERATURE**

Amnioinfusion was first described in 1954, but this technique did not gain popularity till the 1980s. Its use was first reported by Gabbe et al in 1976. He demonstrated in a rhesus monkey that removal of amniotic fluid produced variable deceleration and the replenishment of fluid with saline relieved the deceleration

In 1978 Meis et al evaluated patients with meconium passage in early and late labour and graded the presence of meconium as light or heavy. Heavy meconium passage in early labour was associated with increased fetal morbidity including abnormal fetal heart pattern, prolonged second stage of labour and increased operative interventions. It was also associated with higher incidence of neonatal morbidity and mortality.

Miyazaki and Taylor in 1983 infused saline through intrauterine pressure catheter to women in labour who had either variable deceleration or prolonged deceleration attributed to cord entrapment. They found this improved heart rate pattern in half the patients

Miyazaki and Nevarez in 1985 randomized 96 pregnancies and found multiparous women in labour with cord compression pattern who were treated with amnioinfusion less often required cesarean delivery for fetal distress.

Carson et al in 1976 described combined obstetric and pediatric approach to infants requiring tracheal suctioning at delivery

Wenstrom et al 1989 randomly compared 85 patients with thick meconium. The study group was infused with 1000 ml NS solution initially and every 6 hours until delivery. Control Patients received routine management. The incidence of low 1 minute Apgar score, meconium below the vocal cords and operative delivery were significantly less in patients receiving amnioinfusion. They noted that amnioinfusion is a simple in expensive and safe technique that reduces the incidence of meconium below vocal cords and improve the obstetric outcome in patients in labour with meconium.

Sadovsky et al 1989 demonstrated that 19 cases of labour treated with amnioinfusion for thick meconium had a better neonatal outcome compared with 21 cases treated routinely. The frequency of neonatal acidemia and positive pressure ventilation were significantly reduced in the infused group.

Charles J et al in 1992 studied 170 term and post term patients with thick meconium chosen to receive amnioinfusion or standard obstetric care without amnioinfusion. The rate of fetal distress, caesarean section for fetal distress and rate of meconium aspiration syndrome were significantly reduced in amnioinfusion.

Eriksson and colleagues in 1994 found that amnioinfusion in pregnancy complicated by thick meconium stained amniotic fluid reduced the risk of meconium below the vocal cords and respiratory distress in the newborn.

Lo et al in 1993 and Cialone et al 1994 reported a reduced frequency of acidemia, meconium aspiration syndrome and other neonatal complications in conjunction with amnioinfusion in the setting of thick meconium.

Hofmeyer et al 1998, for the collaborative randomized amnioinfusion for meconium project (CRAMP 1) South Africa did a multi center randomized controlled trial and found that amnioinfusion for meconium stained amniotic fluid reduces the incidence of meconium aspiration syndrome.

Mahomed et al 1998 (CRAMP 2) Zimbabwe found that meconium aspiration syndrome was significantly less frequent in amnioinfusion group and there was trend towards fewer perinatal deaths. In the study there was no difference in the rate of caesarean section.

John pierce et al 2000 did a meta analysis of 13 prospective clinical trials of Intrapartum amnioinfusion for meconium stained fluid. They reached a conclusion that amnioinfusion significantly reduced the frequency of meconium aspiration syndrome, meconium below the vocal cords and neonatal acidemia. The over all caesarean delivery rates were also significantly lower with out increased postpartum endometritis.

Vinita das et al did a prospective case control study in 2001 regarding the safety and efficacy of amnioinfusion during labour complicated by meconium stained liquor and found that amnioinfusion decreases the caesarean rate, incidence of meconium aspiration syndrome and perinatal mortality.

A.M. Rathor et al in 2002 did a randomized controlled trial in 200 women and found that caesarean section rate in amnioinfusion group was less than control group and amnioinfusion was associated with a significant decrease in the incidence of meconium at vocal cords, improvement in 1 minute Apgar score, respiratory distress and fewer admissions to NICU compared with that of controls.

Conchrane database 2002 says amnioinfusion is associated with a reduction in heavy meconium staining of liquor, variable fetal heart decelerations and reduced caesarean section. Also associated with reduction in meconium aspiration syndrome, neonatal hypoxic ischaemic encephalopathy and neonatal ventilation or ICU admission. The database also showed a trend towards reduced perinatal mortality. It concluded that amnioinfusion is associated with improvement in perinatal outcome particularly as settings where facilities for perinatal surveillance are limited.

G.Mukhopadhyay in 2002 did a study to evaluate the safety and efficacy of amnioinfusion and its effect on fetal outcome and found that amnioinfusion decreased the number of abdominal deliveries, decreased the incidence of meconium aspiration syndrome and decreased the admission to neonatal nursery.

N.Talukdar in 2002 found in their study that amnioinfusion decreased the number of neonatal ICU admissions, birth asphyxia and meconium aspiration syndrome.

Abhasingh and Dinesh Magu in 2005 say the use of amnioinfusion has been recognized the most in meconium stained amniotic fluid.

Amnioinfusion is technically feasible in developing countries with limited intrapartum facilities.

## **Fetal Heart Monitoring**

Since the introduction of fetal heart auscultation in the early 19<sup>th</sup> century by Evory Kennedy of Rotunda Hospital, Dublin, the estimation of the FHR has evolved considerably and become the mainstay of intrapartum fetal monitoring. Continuous electronic fetal heart rate monitoring (EFM) was introduced in the late 1960s and since then has attained wide acceptance in clinical obstetrics. During the interpretation of FHR recording, a systematic approach should be used to assess the following aspects.

1. Baseline rate
2. Baseline variability
3. Presence of acceleration
4. Presence of deceleration and their type and severity

A normal FHR trace is one with a baseline rate between 110 and 160 bpm showing good variability (more than 5 bpm) with accelerations and no decelerations. In the presence of normal FHR trace there is only a 2% chance for the fetus to be acidotic ( $\text{pH} < 7.20$ ) and a 1% chance for it to have a 5minute Apgar score  $< 7$  (Beard et al 1971; Steer et al 1989). A definitely 'omnious' or 'pathological' FHR trace is one with a baseline tachycardia ( $> 150$  beats/min) with absent variability and repetitive late or variable decelerations. Other sinister patterns are prolonged severe braducardia ( $< 80$  beats/min) for  $>10$  minutes or a sinusoidal pattern without accelerations.



## **Classification of Intrapartum CTG (FIGO 1987)**

### **1. Normal / Reassuring**

- Baseline heart rate 110-150 bpm
- Baseline variability 5-25 bpm
- Presence of accelerations of at least  $> 15$  bpm lasting  $> 15$  sec
- Absence of decelerations

### **2. Suspicious / equivocal**

- Absence of accelerations for  $> 40$  minute
- Baseline heart rate 150-170 bpm or 100-110 bpm
- Absent baseline variability ( $< 5$ ) for  $> 40$  minutes, with normal baseline and no decelerations
- Variable decelerations  $< 60$  bpm for  $< 60$  sec
- Transient prolonged bradycardia  $< 80$  bpm for  $> 2$  min or  $< 100$  bpm for  $> 3$  min

### **3. Pathological / Ominous**

- Baseline FHR  $> 150$  bpm with absent variability and /or respective late or variable decelerations
- Absent baseline variability ( $< 5$ ) for  $> 90$  min (1 1/2 hrs)
- Complicated variable decelerations
- Prolonged bradycardia ( $< 80$  bpm for  $> 10$  min)
- Sinusoidal pattern with no accelerations

As subacute hypoxia develops during labour, the following FHR changes gradually develop: 1) absence of accelerations 2) gradual increase in baseline heart rate and 3) reduction in baseline variability. The presence of decelerations in the trace indicates the causative mechanism of hypoxia. While variable decelerations suggest cord compression, late decelerations indicate inadequate placental gas exchange.

## **AMNIOINFUSION – GENERAL CONSIDERATIONS:**

### **Definition:**

Amnioinfusion is a procedure in which a physiologic solution such as normal saline is infused into the amniotic cavity.

### **Indications:**

*Diagnostic:*

## **Antepartum**

- Sonographic evaluation of foetal anatomy in oligohydramnios
- To determine the cause of oligohydramnios
  - Fetal anomalies
  - IUGR
  - PROM
- Aspiration of amniofusate for chromosomal study

## **Intrapartum**

- To Know the colour and type of fluid by the regurgitate solution, especially in cases where most of the liquor has been drained

### *Prophylactic:*

#### Intrapartum

- Oligohydramnios
- PPRM
- Prolonged rupture of membranes
- Meconium stained amniotic fluid

### *Therapeutic*

#### Antepartum

- Restore amniotic fluid volume in oligohydramnios
- Prolongation of pregnancy in PROM
- Improvement in FHR variability
- Improvement of odds of success after a failed version

#### Intrapartum

- Variable deceleration
- Chorioamnionitis

In addition to this, repeated trans abdominal amnioinfusion in patients with normally grown fetus but persistent oligohydramnios would prevent pulmonary hypoplasia and compression defects

**Intrapartum amnioinfusion for variable decelerations:**

Many reports have documented relief of variable decelerations with trans vaginal amnioinfusion. Presumably more fluid is associated with less cord compression and thus relieves the variable decelerations, which are the result of cord compression.

Intrapartum amnioinfusion for moderate or thick meconium stained amniotic fluid:

Many randomized studies have reported that amnioinfusion for moderate or thick meconium stained amniotic fluid is associated with a significant decrease in meconium aspiration. For amnioinfusion either RL or NS can be used, the type of solution used has not shown to affect the outcome. These solutions do not cause any electrolyte imbalance in the fetus.

Glantzy and Letteney compared using warmed (37 c) versus room temperature infusion; they found no difference in the outcomes. They also compared using infusion pump versus gravity and found both to be equally effective

**Routes of administration:**

## Transabdominal (antepartum)

Following a prophylactic antibiotic, 20-22 G spinal needle connected to solution through IV tubing is placed in the amniotic cavity under USG guidance. The correct placement is conformed with the loss of resistance and free dispersal of 1-2ml of injected fluid, volume of infusate may vary from 200ml to 300ml depending upon its indication.

## Transcervical (intrapartum)

After spontaneous or artificial rupture of membranes intrauterine catheter (Nelaton catheter) / foleys' catheter /nasogastric tubing is introduced through the dilated cervix beyond the head of the fetus. The catheter is connected to the solution which is infused by gravity or infusion pumps.

Several protocols have been used for amnioinfusion

Amount and type of fluid used in various studies

Wenstrom and Parsons (1989) – 1000 ml NS over 20 – 40 min, repeated every 6 hrs till delivery.

Macri (1992) - 500 ml NS, then 250 ml increment to maintain AFI 10

Cialone (1994) - 600 ml NS for 1<sup>st</sup> hr followed by 150 ml/hr till full dilatation

The protocol used in this study was to infuse 1000 ml of NS as bolus dose over 40 minutes. As per Wenstrom and Parsons study the dose was not repeated because no delivery was delayed for 6 hours in Meconium stained amniotic fluid.

## **Complications due to amnioinfusion:**

### **Maternal**

#### *Systemic:*

- Cardiopulmonary compromise
- Amniotic fluid embolism

#### *Uterine and labour related:*

- PROM
- Preterm labour
- Iatrogenic Polyhydramnios
- Cessarean section scar dehiscence
- Amnionitis

### **Fetal**

- Fetal bradycardia
- Umbilical cord prolapse

Maier et al reported two cases of amniotic fluid embolism in women when an amnioinfusion by infusion pump was ongoing. Several authors have reported the occurrence of excessive uterine contractions or unusually rapid labour progress associated with amnioinfusion.

There is a hypothesis that excessive uterine contractions in some cases are related to extra amniotic placement of amnioinfusion catheter with simultaneous prostaglandin release.

Rapid absorption of the infused fluid could account for the reported cases of pulmonary edema. Consensus of reported data on infectious risk of amnioinfusion suggest that there is no increased risk of maternal and neonatal infection with amnioinfusion.

Owen et al noted decrease in endometritis in women with amnioinfusion compared with those in control.

#### **Amnioinfusion in Meconium stained amniotic fluid :**

Assuming that most of the meconium aspiration occurs at the first neonatal breath, Carson and associates (1976) reported that suction of the fetal nasopharynx immediately after delivery of the head but before the delivery of the body combined with tracheal intubation and suction was effective in reducing the incidence and severity of meconium. But Suresh and Sarkar (1994) noted that despite careful suctioning and lack of neonatal breathing before intubation, 75% still had meconium at trachea.

The current view is that Meconium aspiration occurs due to fetal breathing movements causing inhalation of amniotic fluid with meconium. The breathing which cause inhalation of amniotic fluid are of two types – Gasping and Deep breathing.



**Gasping:**

Gasping is a normal response to hypoxemia and can be induced experimentally by occluding the umbilical cord or by occluding the maternal aorta.

**Deep breathing:**

The fetus may also inhale Meconium by deep irregular breathing in utero, not initiated by hypoxia. These breathes become more frequent as gestation advances and comprise 10% of all fetal breathing movement. Fetal hypercapnoea and acidemia also increase these breathing movements. But these still occur in most of not all normal fetuses.

Thick Meconium is viscid enough to obstruct neonatal airway and concentrated enough to cause significant chemical injury to the tissues. Starks (1980) found that thick Meconium was associated with a higher percentage of low Apgar scores. Mahomed and colleagues (1994) reported that patients with thin Meconium had outcomes indistinguishable from those with clear liquor.

Amnioinfusion dilutes the Meconium and this reduces the toxic effects due to aspiration of thick Meconium. It also increases the volume of fluid around the fetus, thereby decreasing the possibility of fetal distress related to cord compression.

## **PASSAGE OF MECONIUM IN UTERO**

Meconium is found in the fetal gut from 10 weeks' gestation but passage of Meconium is rare before 34 weeks. The incidence of Meconium passage during labour reaches 30% at 40 weeks gestation and 50% at 42 weeks. It is an accumulation of debris that consists of desquamated cells from the alimentary tract and skin, lanugo hairs, fatty material from vernix caseosa, amniotic fluid and various intestinal secretions.

The quantity of Meconium in fetal gut is small during the first two trimesters, but increases rapidly during the third trimester. Because the internal and external anal sphincters are usually closed during fetal life, amniotic fluid ordinarily remains clear. Various stimuli are known to cause relaxation of anal sphincter tone and subsequent passage of Meconium in to amniotic fluid.

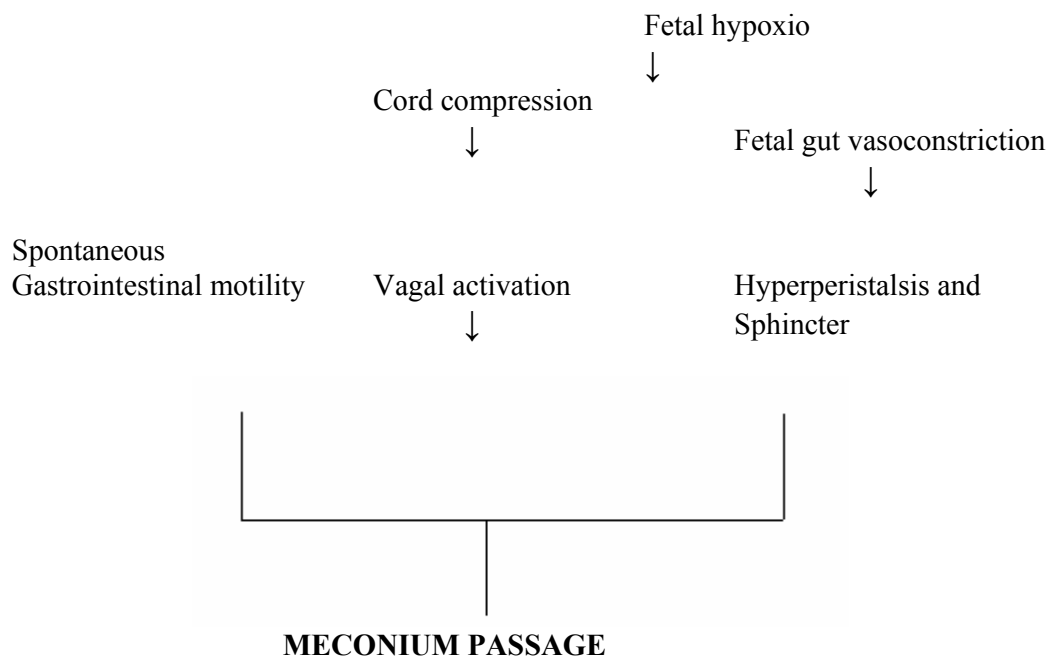
It is suggested that any insult causing fetal hypoxia will cause fetal intestinal hyper peristalsis, relaxation of anal sphincter and thus passage of Meconium. However as Hon suggested parasympathetic stimulation from cord compression may stimulate Meconium passage without concomitant hypoxia.

It is more likely that there is immaturity of the innervation of the fetal gut, with preterm fetuses having fewer myelinated axons and ganglion cells in the colon than term infants. Meconium passage in the preterm fetus can occur if it is infected with organisms which can cause a fetal enteritis eg: *Listeria monocytogenes*, *Ureaplasma urealyticum*, rotaviruses.

It is also possible that Meconium is passed as a result of spontaneous gastrointestinal motility that reflects physiological maturation of fetal gut. As the vast majority of fetuses have no acid base abnormality, the presence of Meconium itself is not a sensitive or specific indicator for the fetal compromise

A number of studies have failed to show any consistent effects of Meconium stained amniotic fluid on Apgar scores, fetal scalp pH or incidents of fetal heart rate abnormalities (Abramnici et al 1974, Miller et al 1975, Baker et al 1992). Moreover one study found that although Meconium stained amniotic fluid correlated poorly with markers of acute intrauterine asphyxia (pH, lactate, Hypoxanthine concentration). It correlated well with blood erythropoietin concentration (marker of chronic asphyxia. Despite these theories, most physicians agree that Meconium stained amniotic fluid in connection with fetal heart rate abnormalities is a marker for fetal distress and is associated with increased perinatal morbidity and mortality

## Aetiology of meconium passage



# Meconium aspiration syndrome

Once Meconium has been passed, regardless of the stimulus, any episode of fetal gasping or respiratory attempts by the new born can cause aspiration of amniotic fluid containing Meconium into the fetal tracheo branchial tree. Meconium aspiration thus occurring can obstruct the airway; interfere with gas exchange and cause severe respiratory distress.

Meconium aspiration syndrome can occur in 10% of cases with Meconium stained amniotic fluid. It comprises a significant range of respiratory compromise. In its mildest form, the disease may present with neonatal tachypnoea associated with normal pH and lower Pco<sub>2</sub>, which resolves in 2 – 3 days. Clinically this mild respiratory morbidity was indistinguishable from transient tachypnoea of newborn. In the more severe form the syndrome can present as severe hypoxemia, acidosis, pneumothorax and respiratory failure a few hours after birth. Pulmonary arterial vasospasm may lead to right to left shunting through patent foramen ovale or ductus arteriosus. Hypoxia further stimulates pulmonary hypertension ultimately leading to convulsions, renal failure, DIC, and heart failure.

Clinically the infants with Meconium aspiration syndrome can have evidence of over inflation of lungs, with a barrel chest. Auscultation can reveal diffuse crepitations and rhonchi. The chest radiograph shows patchy areas of atelectasis and areas of over inflation. Pneumothorax and pneumomediastinum are common – 10 – 20% (Peters and Pendleton 1989). Pleural effusion may be present in about 30%. But

the severity of chest xray abnormalities may not correlate with the severity of clinical disease.

# PATHOPHYSIOLOGY OF MECONIUM ASPIRATION

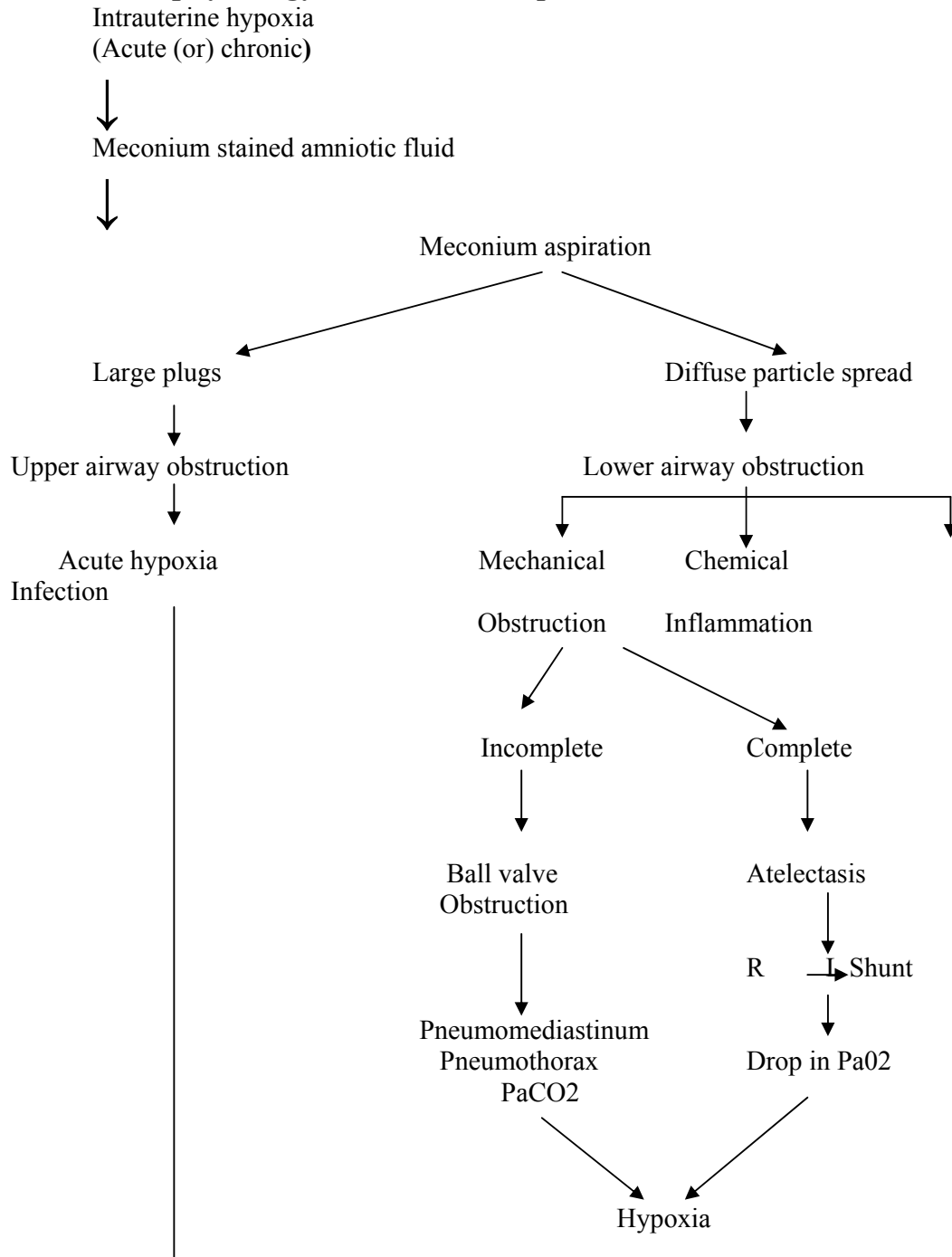
The pathophysiology of Meconium aspiration is extremely complex due to interplay of a large number of mechanisms. Perinatal asphyxia is a critical underlying factor in the pathogenesis of Meconium aspiration syndrome. The pulmonary abnormalities in Meconium aspiration syndrome are related primarily to acute airway obstruction, with obstructive emphysema due to ball valve effect.

Due to the direct irritation and toxicity of Meconium constituents, there is a marked alveolar and parenchymal inflammation and edema with leak of proteins into the airways. There is release of inflammatory mediators like cytokines. Meconium adversely affects the neutrophil function with inhibition of phagocytosis with increased risk of infection. Surfactant dysfunction may occur due to cytotoxic effect on type 2 pneumocytes and decreased level of surfactant protein A and B. There is increased airway resistance and reduced compliance of lungs.

Pulmonary vasoconstriction occurs due to injury of vascular bed of the lung. In these infants, vascular smooth muscle extends into the walls of normally non-muscular intraacinar arterioles and reduces their luminal diameter which subsequently interferes with normal postnatal drop in pulmonary vascular resistance. In addition

these infants may demonstrate plugs of platelets in their small vessels which reduce the cross sectional areas of pulmonary vascular bed.

### Pathophysiology of meconium aspiration





→ Hypercapnia  
Acidosis – Metabolic  
- Respiratory

**Management of infant delivered through Meconium stained amniotic fluid:**

In all cases of Meconium stained amniotic fluid, obstetrician should do the suction of oropharynx, mouth and nose as soon as the head of the baby is delivered.

In 1974, a prospective study by Gregory et al recommended laryngoscopy and tracheal intubation with suctioning for all the newborns born through thick particulate Meconium. In 1976, Carson et al reported a decrease in Meconium aspiration syndrome with suctioning of hypopharynx before the shoulders were delivered and before the first postnatal breathe. They recommended routine tracheal suctioning after as this was not necessary unless Meconium was visible at vocal cords or hypopharynx. Many studies (Wisewell et al; Hageman et al; Stickland et al) have shown beneficial effects of aggressive airway management.

In contrast AAP guidelines of perinatal care recommends endotracheal intubation and direct tracheal suctioning only if there is Meconium at the larynx on laryngoscopy and infant is depressed.

But the study by Wiswell showed that as many as 41 – 54% of infants in whom Meconium aspiration syndrome developed had Apgar score of 8 or grater. Thus a vigorous baby can still develop Meconium aspiration syndrome.

**The current management for any infant delivered with thick Meconium Stained amniotic fluid:**

Place in radiant warmer, immediate intubation and suction of trachea via endotracheal tube with Meconium aspirator.

Repeat the suction till clear, assess for respiratory distress and empty the stomach contents - Meconium stained fluid detected



Notify person skilled in neonatal resuscitation and capable of endotracheal intubation available



Suction mouth and nasopharynx prior to delivery of the shoulders



Place infant on radiant warmer in Trendelenburg



Thick or particulate Meconium - immediately intubate and suction trachea through endotracheal tube with Meconium aspirator. Repeat until clear



Assess for respiratory distress and further management for Meconium aspiration syndrome. Empty the stomach contents.

#### 4. MATERIALS AND METHODS

The study was conducted at Kilpauk medical college hospital, Chennai during the period of September 2004 to August 2005. For this prospective randomized controlled clinical study, 200 women in labour at term with moderate or thick Meconium stained amniotic fluid were included.

# Inclusion criteria

Term pregnancies – Gestational age between 37 and 41 weeks

Singleton pregnancies

Vertex presentation

Normal fetal heart rate and rhythm

Intact membranes at the admittance of the study and having moderate or thick

Meconium stained amniotic fluid on spontaneous rupture of membranes or after doing artificial rupture of membranes in labour ward.

# Exclusion criteria

Multiple pregnancies

Fetal malpresentations

Fetal congenital anomalies

Prelabour rupture of membranes

Polyhydramnios

Antepartum hemorrhage

Medical disease complicating pregnancies – anemia, heart disease, diabetes mellitus, pregnancy induced hypertension

Thin Meconium stained liquor.

Each patient was carefully examined for the presence of any risk factors. Gestational age was confirmed by detailed menstrual history, clinical examination and previous scan report if present.

Abdominal examination was done and fetal presentation determined. Duration and frequency of uterine contractions were assessed. The fetal heart rate was auscultated with pinard fetoscope.

Vaginal examination was done to assess the stage of labour and to confirm the fetal presentation. The amniotic fluid was examined after spontaneous rupture of membranes on artificial rupture of membranes and graded as

Grade i –thin Meconium: having uniform greenish staining and watery

Grade ii – moderate Meconium: having thicker greenish staining but still watery

Grade iii – thick Meconium: green with particulate matter having pea soup quality (thick tenacious opaque)

These patients were categorized into amnioinfusion group and no amnioinfusion group in a 1:1 ratio. The first patient was given amnioinfusion whereas second patient was taken as control group (without amnioinfusion). The procedure was explained to the patient and consent obtained.

NST tracing was taken soon after the rupture of membranes. In the amnioinfusion group the patient was asked to lie in dorsal position and Foleys catheter was introduced transcervically under aseptic precautions such that its tip lies just above the baby's head. Normal saline at room temperature was infused through the catheter using intravenous tube.

The amnioinfusion was given as 1000 ml of normal saline over 40 minutes. The control group did not receive any catheter placement. Both groups were managed in the labour ward routinely.

In the amnioinfusion group the NST tracing was repeated after giving amnioinfusion. In control group the NST tracing was repeated after 1 hour.

Uterine tone and frequency of contraction were monitored by abdominal palpation. Oxytocin drip was started if contractions were not effective. Fetal heart rate was auscultated every 15 minutes during the first stage of labour and every 5 minutes during the second stage of labour using pinard fetoscope. Fetal distress was defined as fetal bradycardia(<120 beats/min) or fetal tachycardia(>160 beats/minute).

Progress of the labour was monitored and obstetric assistance was given whenever necessary. Forceps application and delivery by caesarean section were done for fetal heart rate abnormalities or failure to progress such as arrest disorders or protraction disorder as indicated.

During delivery as soon as baby's head was delivered, the eyes were cleaned and suction was applied to the oropharynx, mouth and nose before the delivery of the shoulders by the obstetrician. Any mucus or Meconium was aspirated. Paediatrician attended all the newborns immediately. After suctioning of oropharynx and nasopharynx, laryngoscopic visualization of the vocal cords was done by the paediatrician. The presence or absence of Meconium below the vocal cords were evaluated and documented. The staining of baby with Meconium and one minute, five minute Apgar were observed and recorded. If the baby had respiratory distress or five minute Apgar score less than 6, then baby was taken to NICU and treated accordingly.

After delivery patients were observed in labour ward for two hours and then shifted to postnatal ward. Babies were followed prospectively till the day of discharge from the hospital



## 5. RESULTS AND ANALYSIS OF THE STUDY:

**Table 1: Age**

Age (in years)	Amnioinfusion Group	Control Group
< 20	31	22
21 – 25	42	53
26 – 30	22	16
> 30	5	9

P Value :  $0.17 > 0.05$  – not significant

In this study the age of the amnioinfusion group is matching with the control group.

**Table 2: Parity**

Parity	Amnioinfusion Group	Control Group
Nulli para	67	58
Para 1	24	31
Para 2	8	10
Para 3	1	1

P Value =  $0.62 > 0.05$  – not significant

In this study parity of both the groups were matching. In both the groups nulliparas were predominant.

**Table 3: Socio economic status**

Socio economic status	Amnioinfusion Group	Control Group
1	0	0
2	0	0
3	0	0
4	42	40
5	58	60

P Value = 1 > 0.05 – not significant

In this study socio economic status of amnioinfusion group matching with the control group. In both groups socio economic status 4&5 were predominant.

**Table 4: Gestational Age**

Gestational Age ( in weeks)	Amnioinfusion Group	Control Group
37 – 39	79	72
40 – 41	21	28

P Value = 0.249 > 0.05 – not significant

In this study both groups were matching in relation to their gestational age. Most of the patients were between 37 to 39 weeks.

**Table 5: Phase of labour in inclusion**

Phase of labour on inclusion	Amnioinfusion Group	Control Group
Active Phase	85	83
Latent phase	15	17

P Value =  $0.847 > 0.05$  – not significant

In amnioinfusion group 85 patients were in the active phase of labour compared to 83 in the control group. In control group 17 patients were in the latent phase of labour compared to 15 patients in amnioinfusion group. Both the groups were matching with the phase of labour in inclusion.

**Table 6 : Oxytocin augmentation**

Oxytocin augmentation	Amnioinfusion Group	Control Group
Given	64	67
Not Given	36	33

P Value =  $0.726 > 0.05$  – not significant

Oxytocin augmentation was required in 64 patients in amnioinfusion group and 67 patients in control group. Both the groups were matching with the oxytocin augmentation requirement.

**Table 7: Intrapartam FHR Abnormalities**

Fetal Heart Abnormalities	Amnioinfusion Group (After giving Amnioinfusion)	Control Group
Present	24	38
Not Present	76	62

P Value < 0.05 – significant

There were more cases of fetal distress in the control group (38) compared with the amnioinfusion group (24). P Value is < 0.05. This shows that amnioinfusion causes a significant reduction in fetal heart rate abnormality during labour.

**Table 8: Types of FHR Abnormalities in Amnioinfusion Group**

Types of Fetal Heart Abnormalities	Before giving Amnioinfusion	After giving Amnioinfusion
Tachycardia	8	8
Bradycardia	5	5
Loss of beat to beat variability	7	7
Variable deceleration	14	4
Total	34	24

**Table 9: Types of FHR Abnormalities in Control Group**

Types of Fetal Heart Abnormalities	First NST	Second NST (after 1 hour)
Tachycardia	7	7
Bradycardia	10	10
Loss of beat to beat variability	7	7
Variable deceleration	14	14
Total	38	38

Amnioinfusion significantly reduces the incidence of variable deceleration

In control group there is no change in both NST tracings

**Table 10: Mode of Delivery**

Mode of delivery	Amnioinfusion Group	Control Group
L N	61	51
Out let	2	6
L M C	3	4
Vacuum	0	1
LSCS	34	38

P Value = 0.1859 > 0.05 – not significant

When mode of delivery is considered 61 in amnioinfusion group and 51 in control group delivered by labour natural. 2 patients in amnioinfusion group and six patients in control group had outlet forceps delivery. Low mid cavity forceps used in 3 patients belonging to the amnioinfusion group and 4 patients in the control group. Vacuum forceps was needed in 1 patient in control group and none in the amnioinfusion group. 34 patients in the amnioinfusion group and 38patients in the control group were delivered by LSCS. This shows that amnioinfusion is not influencing the mode of delivery.

**Table 11: Incidence of Instrumental Delivery**

Deliveries	Amnioinfusion Group	Control Group
Instrumental	39	49
L N	61	51

P Value = < 0.05 – significant

This study 39 patients of amnioinfusion group and 49 patients of control group were needed instrumental delivery. Where as 61 patients in amnioinfusion group and 51 patients were delivered by labour natural.

**Table 12: Indication for instrumental deliveries**

Indication	Instrumental Delivery	Amnioinfusion Group	Control Group
Fetal Distress	LSCS	21	34
	Forceps	3	4
	<b>Total</b>	<b>24</b>	<b>38</b>
Others	LSCS	13	4
	Forceps	2	6
	<b>Total</b>	<b>15</b>	<b>10</b>

P Value = < 0.05 – significant

Fetal distress was the indication for instrumental delivery in 24 patients of amnioinfusion group and 38 patients in control group. Where as other causes were the indication for instrumental delivery in 15 patients of amnioinfusion group and 10 patients in control group and one patient in control group delivered by vacuum. Amnioinfusion decreases the incidence of fetal distress.

**Table 13 : One minute Apgar**

One Minute Apgar	Amnioinfusion Group	Control Group
0 – 4	3	10
5 – 7	76	89
8 – 10	21	1

P Value : -  $0.00 < 0.05$  – Significant

One minute apgar less than 4 was seen in 3 infants in amnioinfusion group and 10 infants in control group, 5 – 7 was seen in 76 infants in amnioinfusion group and 89 infants in control group. 8 – 10 apgar score was seen in 21 infants in amnioinfusion group and one infant in control group.

**Table 14 : 5 minute Apgar**

5 minute Apgar	Amnioinfusion Group	Control Group
0 – 4	0	3
5 – 7	9	48
8 – 10	91	49

P Value :  $0.00 < 0.05$  - Significant

Five minute apgar score more than 7 was seen in 91 infants in amnioinfusion group and 49 infants in control group. Both one minute and five minute apgar score were significantly raised in amnioinfusion group.



**Table 15 : Baby weight**

Baby Weight ( in Kgs)	Amnioinfusion Group	Control Group
2 – 2.5	29	28
2.6 – 3	40	32
> 3	31	40

P Value :  $0.35 > 0.05$  – Not Significant

In this study, birth weight of the babies of the amnioinfusion group matching with the control group.

**Table 16 : Grading of Meconium in Inclusion**

Meconium Grading	Amnioinfusion Group	Control Group
Moderate	75	78
Thick	25	22

P Value :  $0.18 > 0.05$  - not Significant

In this study, Grading of the meconium in inclusion of the amnioinfusion group matching with the control group.

**Table 17 : Meconium below vocal cord**

Meconium below vocal cord	Amnioinfusion Group	Control Group
Present	10	27
Absent	90	73

P Value :  $0.0035 < 0.05$  - Significant

In this study the presence of meconium below the vocal cord was significantly raised in control group.

**Table 18 : Incidence of Meconium aspiration syndrome**

Meconium aspiration syndrome	Amnioinfusion Group	Control Group
	3	14

P Value :  $0.00 < 0.05$  – Significant

The incidence of meconium aspiration syndrome in amnioinfusion group 3 in number (presence of meconium below the vocal cord were in 10 patients) where as 14 in number of control group. (presence of meconium below the vocal cord were in 27 patients). This is significant.

**Table 19 : Admission in NICU**

Admission in NICU	Amnioinfusion Group	Control Group
Admission	13	31
No Admission	87	69

P Value :  $0.00 < 0.05$  – Significant

13 neonates of amnioinfusion group and 31 neonates of control group were needed admission in NICU. Amnioinfusion reduces the need of admission in NICU.

**Table 20 : Reasons for Neonatal admission**

Reason	Amnioinfusion Group	Control Group
Observation	7	13
MAS	3	14
Birth Asphyxia	3	4

P Value :  $0.00 < 0.05$  – Significant

7 neonates in the amnioinfusion group and 13 neonates in the control group were admitted for observation. 3 neonates of the amnioinfusion group and 4 of the control group were admitted for birth asphyxia, where as 14 neonates of the control group and 3 of the amnioinfusion group were admitted for Meconium Aspiration Syndrome. Meconium aspiration syndrome is the predominant cause for the NICU admission in the control group.

**Table 21 : Perinatal morbidity and mortality**

Perinatal Outcome	Amnioinfusion Group	Control Group
Perinatal Morbidity	13	31
Perinatal Mortality	0/100	2/100

P Value :  $0.00 < 0.05$  – Significant

Perinatal morbidity and mortality were increase in the control group.

Amnioinfusion decrease the perinatal morbidity and morality.

**Table 22 : Nature of delivery in Latent phase**

Nature of delivery	Amnioinfusion Group	Control Group
LSCS	10	12
LMC	1	0
LN	4	5
<b>Total</b>	<b>15</b>	<b>17</b>

P Value :  $> 0.05$  – not Significant

Nature of the delivery in the amnioinfusion group was matching with the control group.

## **6. DISCUSSION**

In this prospective study, patients with moderate and thick Meconium stained amniotic fluid, 100 patients received amnioinfusion and 100 patients did not receive amnioinfusion.

The amnioinfusion group and control group were matched with respective to age, parity and gestational age.

More patients in both the groups were in the active phase of labour on inclusion than latent phase but this difference was not significant.

The number of patients receiving oxytocin for augmentation is not influenced by amnioinfusion (64% versus 67% - P value > 0.05)

But in the study by Vsta et al, the amnioinfusion group had significantly higher incidence of oxytocin use than the control group ( 44% versus 23% - P value < 0.001). The study of Cialone et al also showed the patient receiving amnioinfusion had greater oxytocin requirement in labour.

In this study there is no significant difference between the two groups in the rupture of membrane to delivery interval (2.44 hrs versus 2.24 hrs). This is correlating with the study of Cialone et al in which the rupture of membrane to delivery interval was similar in both groups. Vsta et al showed longer interval in the amnioinfusion group.

The incidence of fetal distress in this study was 24% in the amnioinfusion group and 38% in the control group (  $P < 0.05$ ). This is statistically significant. This is in par with Wenstrom and Parson study which showed decrease in the incidence of fetal distress in the amnioinfused patients. But in contrast study by Rogers et al showed a higher incidence of fetal distress in the amnioinfused group. (30.5% versus 19.7 %). Ericson et al also demonstrated that the incidence of fetal distress was not significantly different between the two groups, which is in contrast to this study.

Taking into account on the phase of labour on inclusion most of the patients in latent phase of labour delivered by caesarean section while most of the patients in the active phase of labour on inclusion delivered vaginally.

There was no significant difference between the two groups regarding the mode of delivery. Results of other study on the effect of intrapartum amnioinfusion on caesarean section rates are given in the table below (Table A). The last three studies mentioned in the table show a reduction in the caesarean section rates with the amnioinfusion while other studies do not show a significant difference.

In this study the number of operative deliveries for fetal distress were significantly lower than that in control group ( 24% versus 38% -  $P \text{ Value} < 0.05$ ). This is similar to the result in the study by A.M. Rathore et al (12% versus 26%). Lo and Rogers, Hoodley et al and Marci et al also reported similar findings. Ericson et al and Sponge et al reported no effect on caesarean section for fetal distress. Vsta et al reported a higher incidence of caesarean delivery in the amnioinfusion group (28% versus 17%)

especially for fetal distress (16% versus 11%). The CRAMP 1 & 2 also showed no significant difference in the caesarean section rates for fetal distress between the two groups.

In this study, the one minute Apgar score  $< 4$  was 3% versus 10% (P value  $< 0.05$ ).

One minute Apgar score 5 – 7 was 76% versus 89% ( P value  $< 0.05$ ) and

One minute Apgar score 8 – 10 was 21% versus 1% (P value  $< 0.05$ ).

5 minute Apgar score

$< 7$  was 9% versus 48% - P value  $< 0.05$

8 – 10 was 91% versus 49% - P value  $< 0.05$

Hence the fetal out come was good with the amnioinfusion group than the control group. A.M.Rathore et al reported similar findings in their study. The studies by Wenstrom and Passon, Marci et al, Keith and Rogers et al showed a significant decrease number of babies with low Apgar in the amnioinfusion group.

In this study, the presence of meconium below the level of vocal cord is significantly reduced in the amnioinfusion group. ( 10% versus 27% - P value  $< 0.05$ ). The incidence in the other studies given in table B. All the studies show a reduction in the incidence of meconium below the level of vocal cord with amnioinfusion except that of Sponge et al and Vsta et al.

The incidence of Meconium Aspiration Syndrome in this study 3 versus 14 ( P value < 0.05). This shows that there is significant decrease in the incidence of meconium aspiration syndrome in the amnioinfusion group. Other studies showing similar results are given in table C. All the studies show decrease in the incidence of meconium aspiration syndrome with amnioinfusion.

In this study 13 neonates from the amnioinfusion group and 31 from the control group needed admission in the NICU. This shows that there is a significant decrease in the neonatal admissions in the amnioinfusion group (13% versus 31% - p value < 0.05). CRAMP 2 Zimbabwe arm showed similar results (12.8% versus 22.9%). The study by A.M.Rathore et al also showed decreased neonatal admissions in the amnioinfusion group (3% versus 11%). Studies by Erikssen et al, Cialone et al and CRAMP 1 South African arm also showed similar results.

In this study, the perinatal mortality rate in the amnioinfusion group is 0% while in the control group is 2%. The difference is not significant statistically. In the CRAMP 2 study conducted in Zimbabwe the perinatal mortality was 1.2% in amnioinfusion group versus 3.6% in the control group. In the study by A.M.Rathore et al the perinatal mortality rate was 2% in the amnioinfusion group and 5% in the control group. G.Mukhopadhyay showed a perinatal mortality rate of 2% in the amnioinfusion group and 11% in the control group.

In this study, no adverse effects were noted in the fetus or mother due to amnioinfusion.



**Table A: Intrapartum amnioinfusion and LSCS rates**

Study	Amnioinfusion Groups (%)	Control Groups (%)
Spong et al – 1994	8 / 43 (18.6)	9 / 50 (18)
Galon et al – 1994	14 / 47 (29.8)	11 / 58 (19)
Hofmeyr et al – 1998	70 / 167 (41.9)	68/ 159 (42.8)
Mohamed et al – 1998	30 / 317 (9.5)	37 / 328 (11.3.)
Moodley et al –1998	3 / 30 (10)	7 / 30 (23)
Khosia et al - 1997	1 / 25 (4)	7/25 (28)
A M Rathore et al – 2002	21 / 100 (21)	36 /100 (36)
Present Study	34 / 100 (34)	38 / 100 (38)

**Table B: Intrapartum Amnioinfusion and presence of Meconium below the vocal cord**

Study	Amnioinfusion Groups (%)	Control Groups (%)
Sadorsky et al 1989	0 / 19 (0.0)	6 / 21 (28.6)
Macri et al 1992	4 / 85 (4.7)	33 / 85 (38.8)
Sprg et al 1994	3 / 43 (7.0)	2 / 50 (4.0)
Gialong et al 1994	2 / 47 (4.2)	34 / 58 ( 58.6)
Ericson et al 1994	1 / 65 (1.5)	8 / 59 (13.6)
USK et al 1995	28 / 97 (29.0)	31 / 112 (28.0)
Hofmeyr et al 1998	6 / 158 (3.8)	12 / 164 (7.3)
Khosh et al 1997	3 / 25 (12.0)	4/125 ( 16.0)
A. M Rathore et al 2002	10 / 100 (10.0)	24 / 100 (24)
Present Study	10 /100 (10.0)	29 / 100 (29.0)

**Table C: Intrapartum Amnioinfusion and Meconium Aspiration Syndrome**

Study	Amnioinfusion Groups (%)	Control Groups (%)
Wenstrom et al 1989	0 / 36 (0.0)	3 / 44 (6.8)
Macri et al 1992	0 / 85 (0. 0)	5 / 85 ( 5.9)
Lo and Rogess et al 1993	1 / 60 (1.7)	3 / 52 (5.8)
Cialna et al 1994	1 / 47(2.1)	6 / 58 (13.6)
Ericson et al 1994	0 / 65 (0.0)	2 / 59 (3.4)
USK et al 1995	19 / 497 (3.8)	20 / 440 (4.5)
Mohamed et al 1998	10 / 323 (3.1)	42 / 329 (12.8)
A. M Rathore et al 2002	0 / 100 (10.0)	1 / 100 (1.0)
Present Study	3 / 100 (3.0)	14 / 100 (14.0)

## **7. SUMMARY**

In this prospective case controlled study, 200 patients with moderate or thick meconium stained amniotic fluid were selected from labour ward. Among them 100 were given Amnioinfusion and grouped as amnioinfusion group. The other 100 were kept as control group with out giving amnioinfusion.

In analyzing the intrapartum course and perinatal outcome, the following points were summed up:

1. Majority of patients in amnioinfusion group and control group were in the age group of 19-30. Only 5% in amnioinfusion group and 9% in the control group were above 30
2. The parity is also same in both the groups. Majority of the patients were nullipara - 67% in amnioinfusion group and 58% in control group.
3. The gestation age for majority of the patients in the amnioinfusion group and control group were between 37 to 40 weeks. Only 21% of the amnioinfusion group and 28% in the control group had gestational age above 40 weeks.
4. On inclusion majority of the patients were in the active phase of labour. 85% in amnioinfusion and 83% in the control group
5. Augmentation with oxytocin was needed in 64% patients in the amnioinfusion and 67% in control group.

6. The mean duration of labour from rupture of membrane to delivery was 2.44 hrs in the Amnioinfusion group and 2.24 hrs in control group. The difference is not significant and this shows that in this study there was no effect on the duration of labour by Amnioinfusion.
7. The incidence of FHR abnormalities was significantly lower in the Amnioinfusion group, 24% versus 38% in the control group.
8. Considering the mode of delivery, majority of the patients in both the groups delivered by L N 61% in Amnioinfusion group and 51% in control group. The incidence of instrumental vaginal delivery was 5% in amnioinfusion group and 11% in control group.
9. The number of patients who delivered by LSCS was 34% in Amnioinfusion and 38% in control group which is not significant.
10. There was significant decrease in the number of LSCS done for fetal distress in Amnioinfusion group, 21% versus 34% in control group.
11. Most of the patients who were included in the study in the latent phase of labour underwent LSCS.
12. Discussing about the perinatal outcome, there was significant decrease in the number of babies with low Apgar score.
13. The presence of meconium below the vocal cords was significantly reduced in the amnioinfusion group, 10% versus 27% in the control group.

14. The incidence of meconium aspiration syndrome was also significantly reduced in amnioinfusion group, 3 versus 14 in control group. This shows that amnioinfusion is effective in reducing meconium below vocal cord and meconium aspiration syndrome.
15. New born babies admitted in NICU was lower in amnioinfusion group than the control group (13% versus 31%). Hence there is significant decrease in the neonatal admission in the amnioinfusion group.
16. Perinatal mortality in amnioinfusion group 0% where as in control 2%.
17. There are no adverse maternal or fetal complications reported with amnioinfusion in this study.

## 8. CONCLUSION

In this study it is found that the use of amnioinfusion has significantly reduced the number of intrapartum fetal heart rate abnormalities.

Amnioinfusion has no effect on the mode of delivery in patients with meconium strained amniotic fluid in labour. The incidence of caesarean deliveries is not reduced but the number of caesarean section done for fetal distress has been decreased by the use of amnioinfusion.

The study shows a drastic improvement in the perinatal outcome in the patients who received amnioinfusion. There was significant improvement in the one minute and five minute Apgar score, decrease in the incidence of meconium below the level of vocal cord and decrease in meconium aspiration syndrome. The number of babies admitted in NICU is also significantly reduced in the amnioinfusion group.

To conclude, amnioinfusion is a safe, easy and effective procedure, which needs little expertise and can be done in labour ward even with limited facilities to improve neonatal outcome in meconium stained liquor. The use of such simple technique is very useful and effective in developing countries even with limited newborn care facilities. It is also very cost effective.

## 9. PROFORMA

Name :                      Age :                      I.P. No.:                      DOA:

DOD:

Gravida	Para	LMP
1	0	10/1/1998
2	1	10/1/1998
3	2	10/1/1998
4	3	10/1/1998
5	4	10/1/1998
6	5	10/1/1998
7	6	10/1/1998
8	7	10/1/1998
9	8	10/1/1998
10	9	10/1/1998
11	10	10/1/1998
12	11	10/1/1998
13	12	10/1/1998
14	13	10/1/1998
15	14	10/1/1998
16	15	10/1/1998
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91	90	10/1/1998
92	91	10/1/1998
93	92	10/1/1998
94	93	10/1/1998
95	94	10/1/1998
96	95	10/1/1998
97	96	10/1/1998
98	97	10/1/1998
99	98	10/1/1998
100	99	10/1/1998

EDD

Booked / Unbooked

### Socio-economic status

### Gestational Age

### Duration of First Stage of Labour

### Duration of Second Stage of Labour

Colour of Liquar when membrane ruptured – Meconuim	Moderate
--	----------

Thick

Time Interval between rupture of membrane to delivery

## General Examination

Nutrition	Height
-----------	--------

Anemia	Weight
--------	--------

## Pedal Edema

Vital Signs - P.R

B.P

Temp

CVS :

RS :

P/A :

P/V :

## Investigations

Urine - Alb

Blood Group and Rh type

Sugar

Deposits

Hb



NST

Baseline FHR

Beat to beat Variability

Acceleration

Deceleration -

Amnioinfusion : Given / Not Given

Repeat NST

Baseline FHR

Beat to beat Variability

Acceleration

Deceleration -

Oxytocin Augmentation - Given / Not Given

Mode of Delivery

Labour Natural

Forceps

LSCS Indication

Baby

TOD

Birth Weight

Sex

Apgar

1 Minute

5 Minute

Resuscitation Method

Condition of Baby - Admission

No Admission

Meconium Below the vocal cord

Meconium Aspiration Syndrome

Other Neonatal Complication

Neonatal mortality and cause

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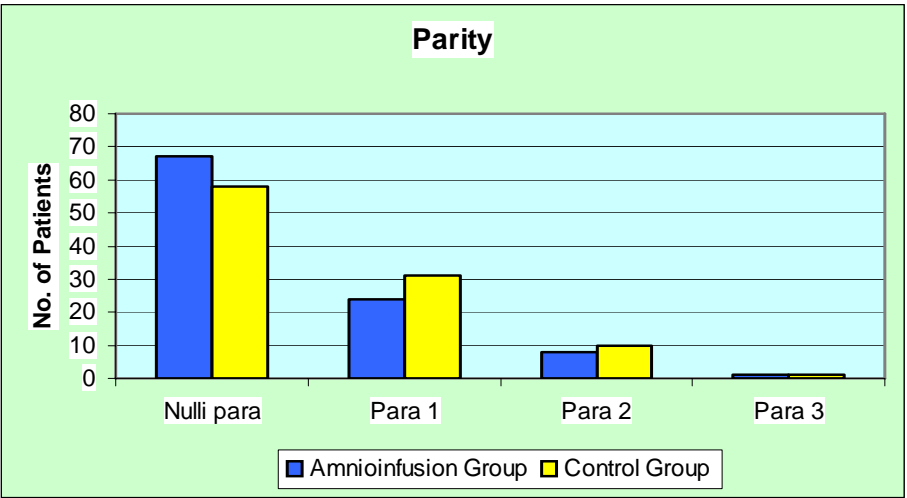
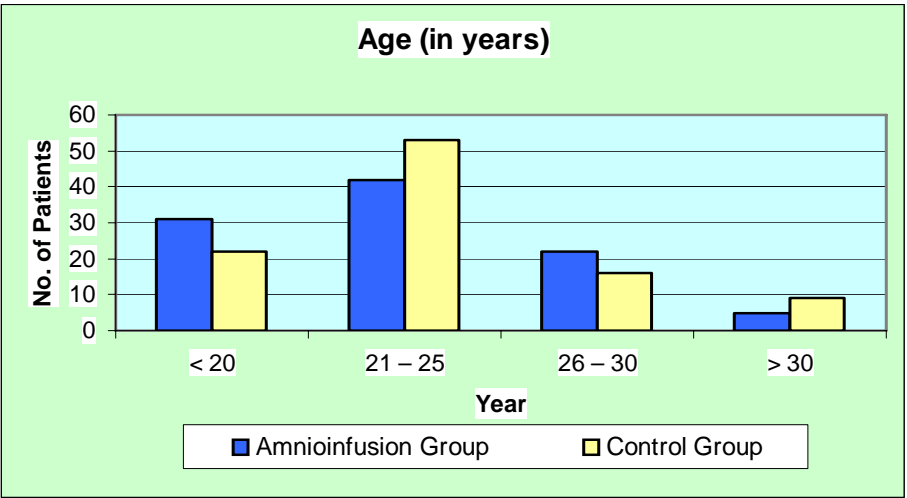
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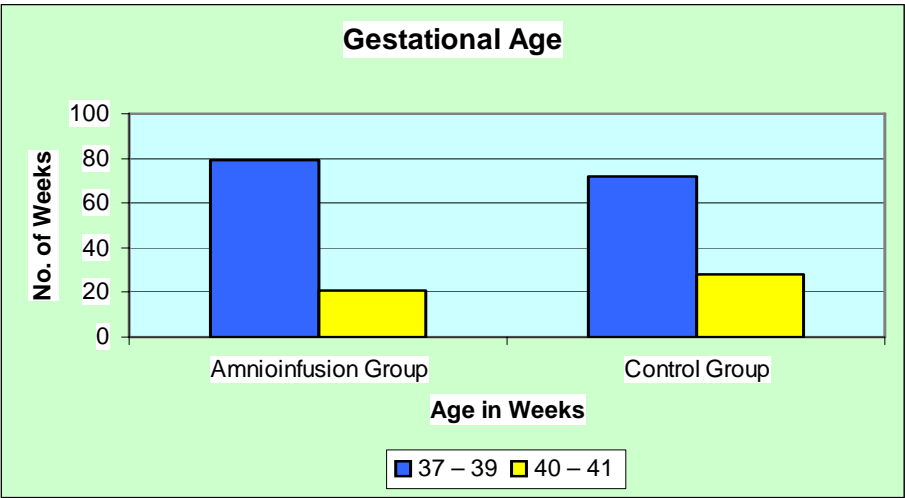
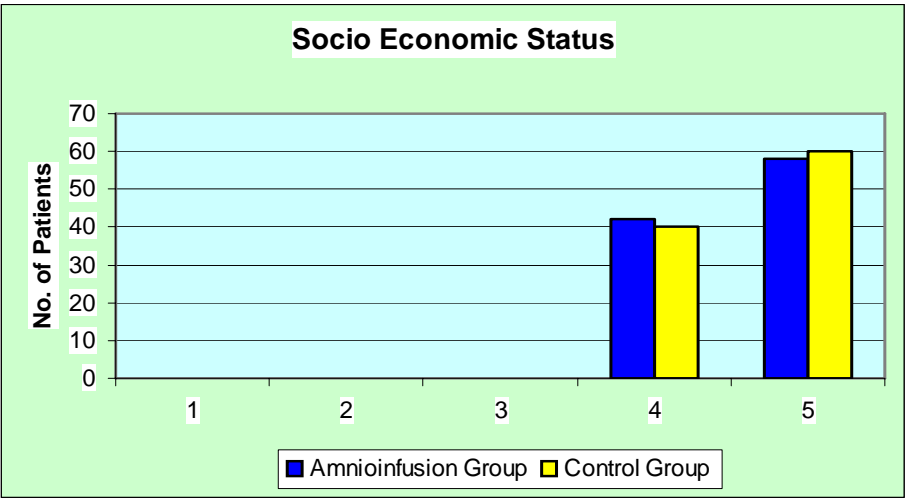
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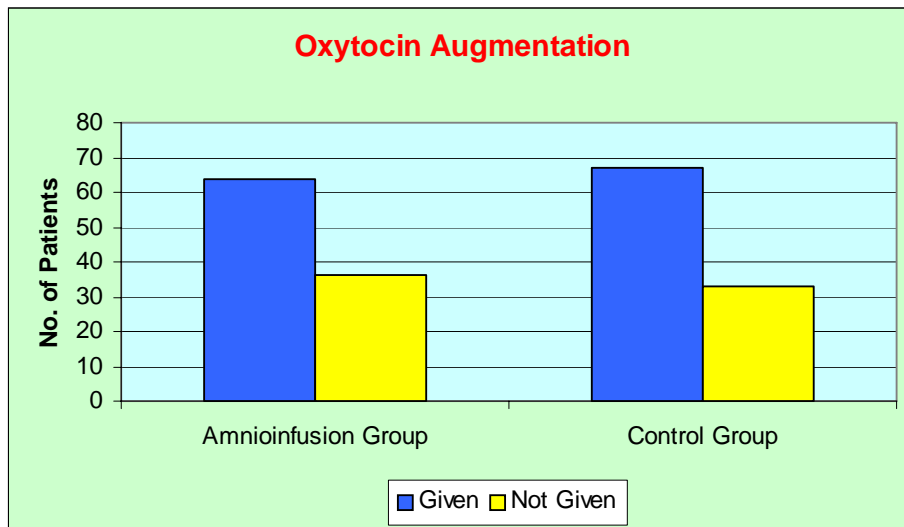
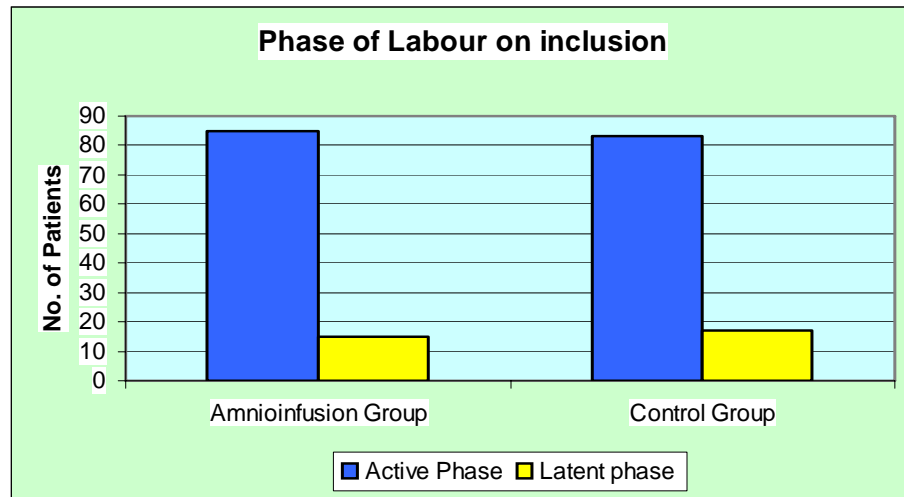
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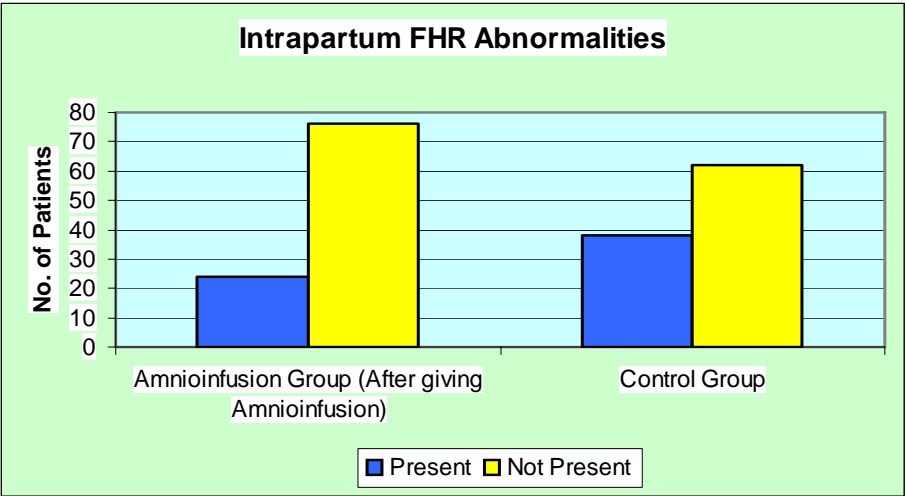
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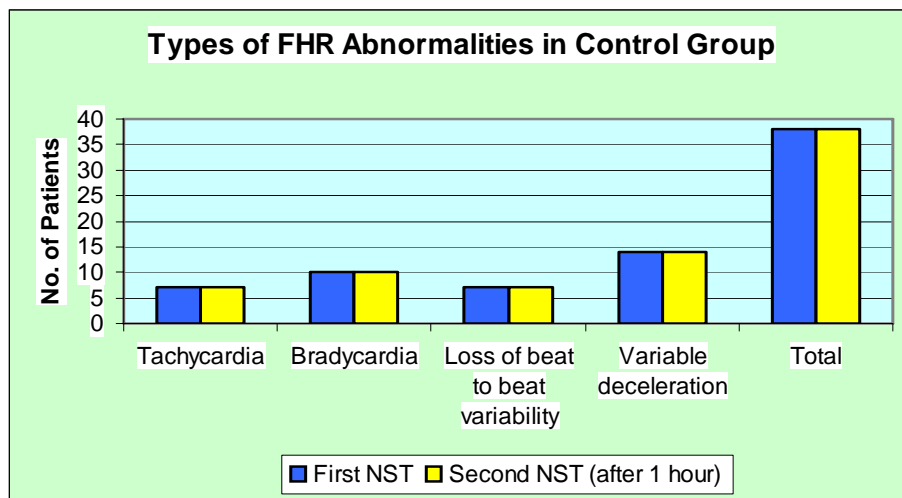
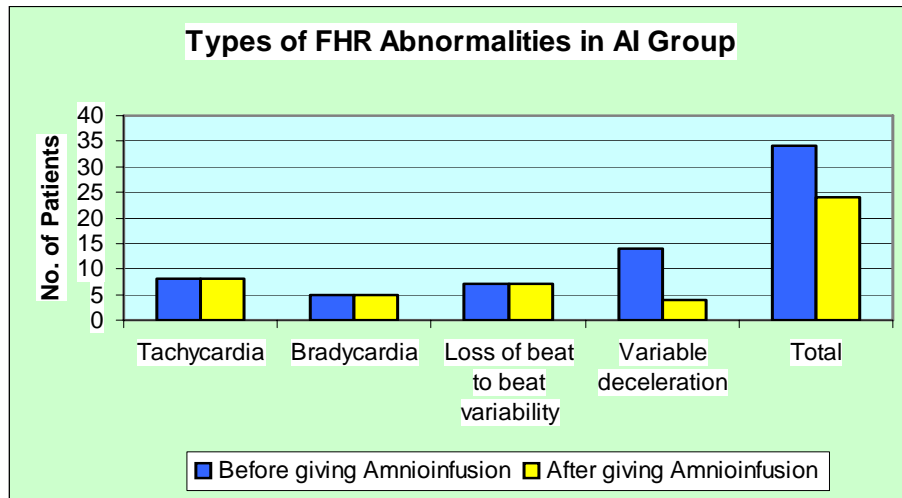


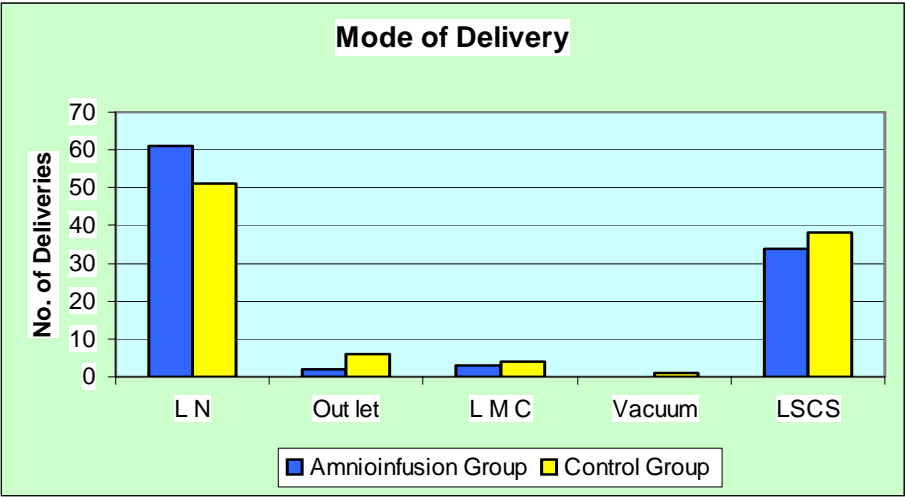


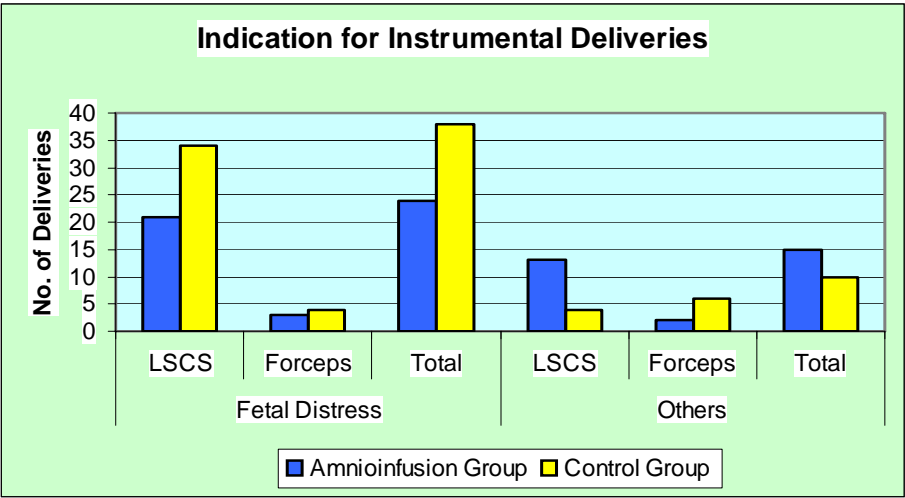
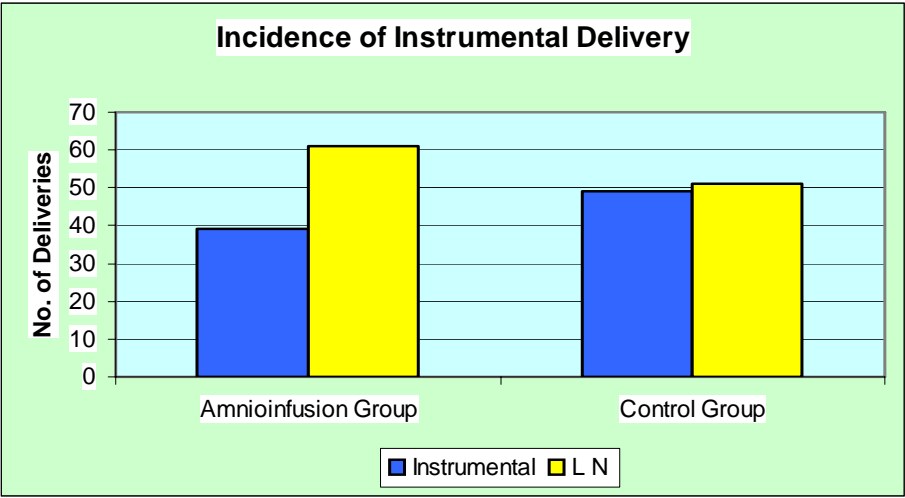


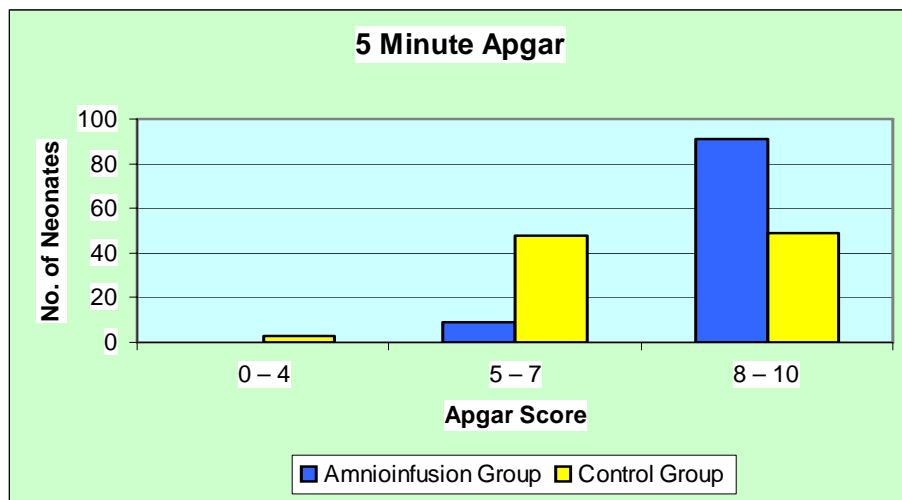
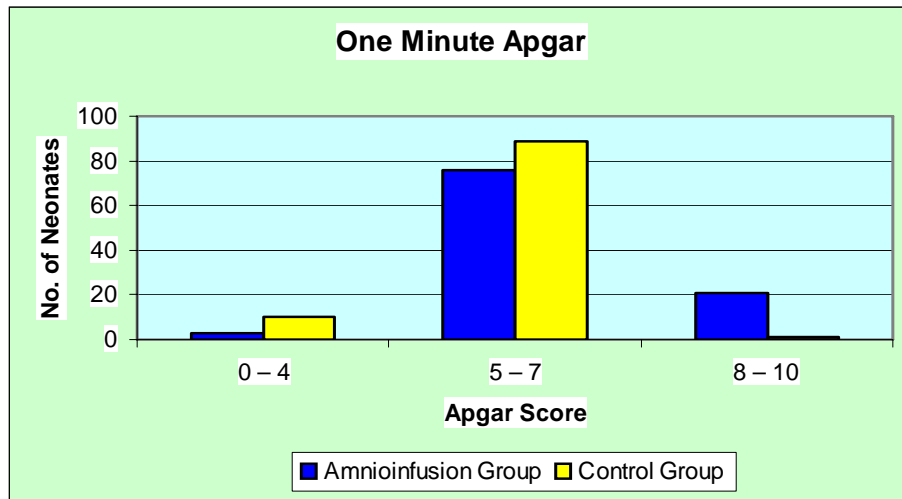


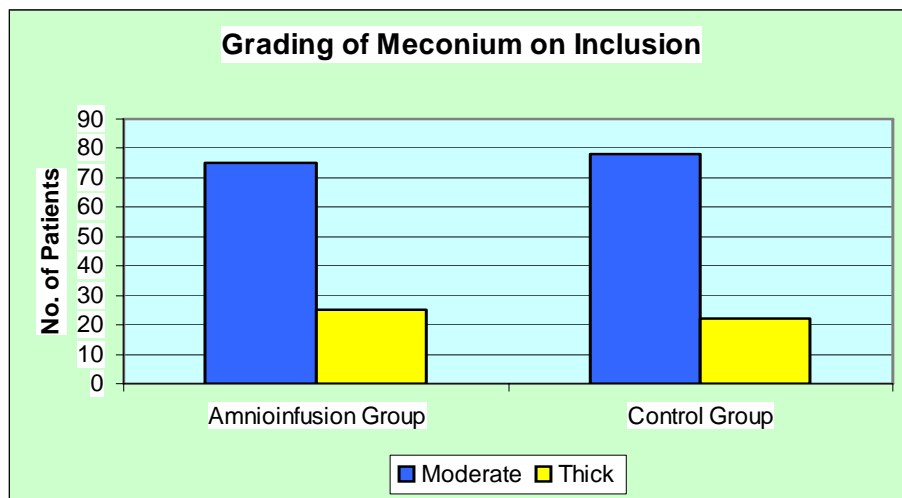
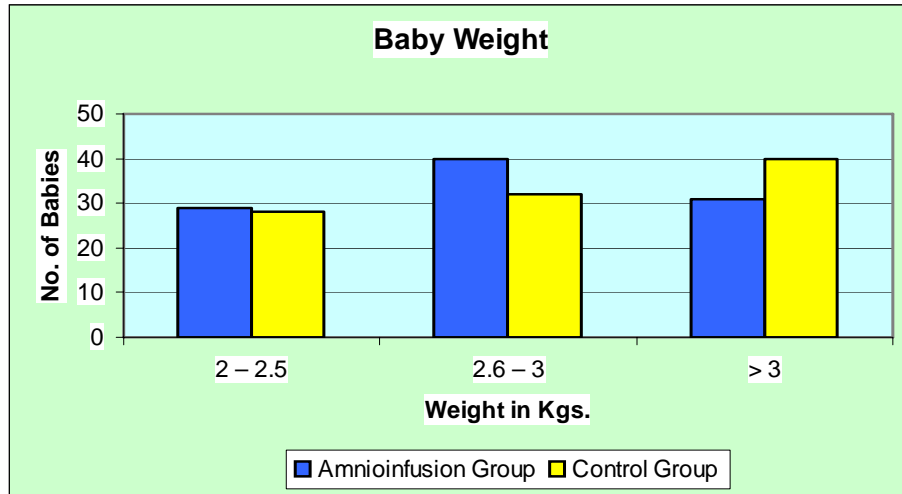


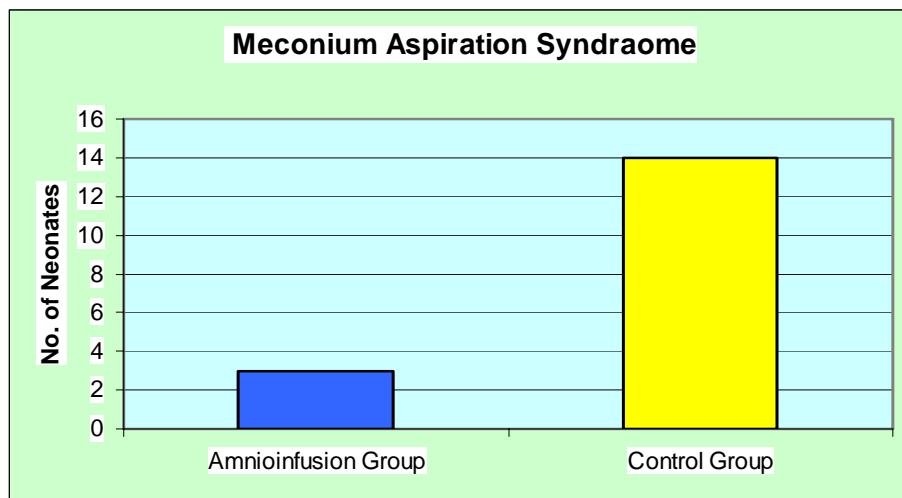
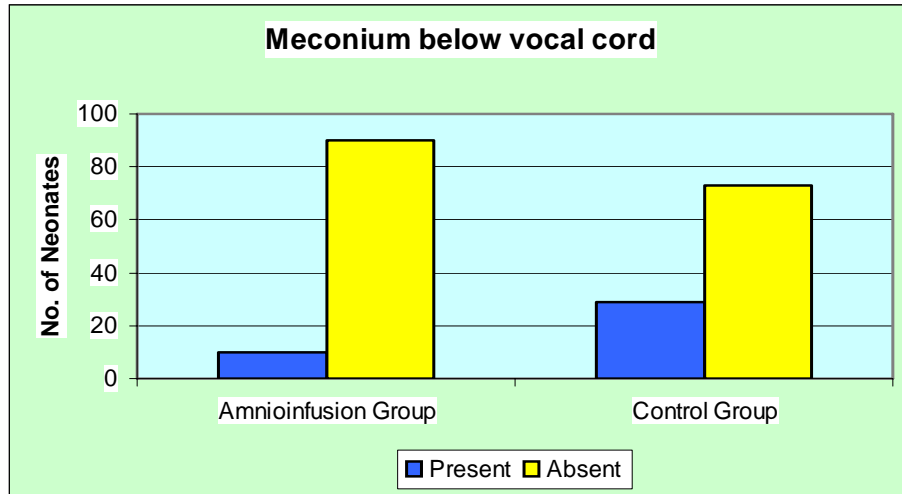




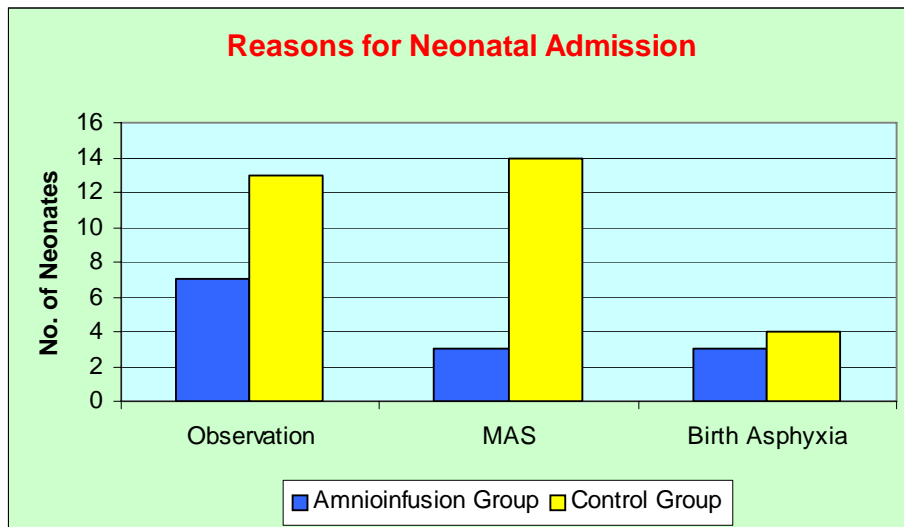
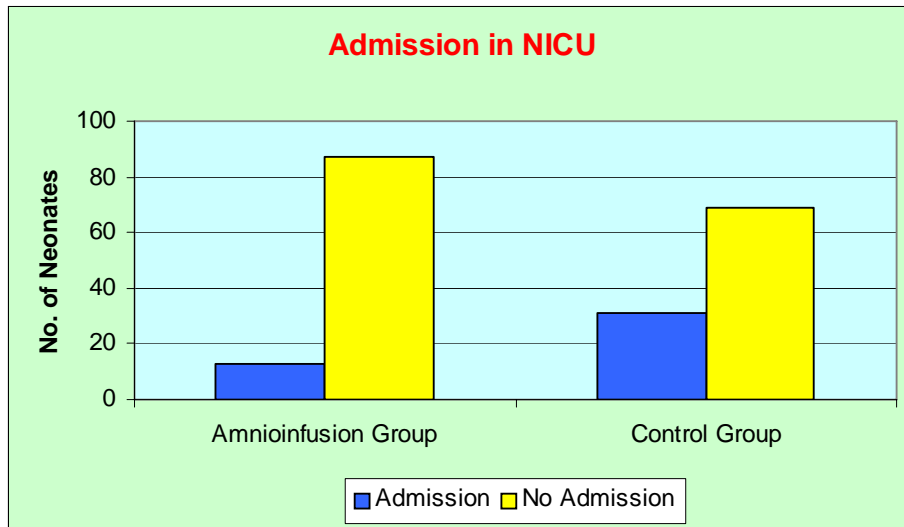


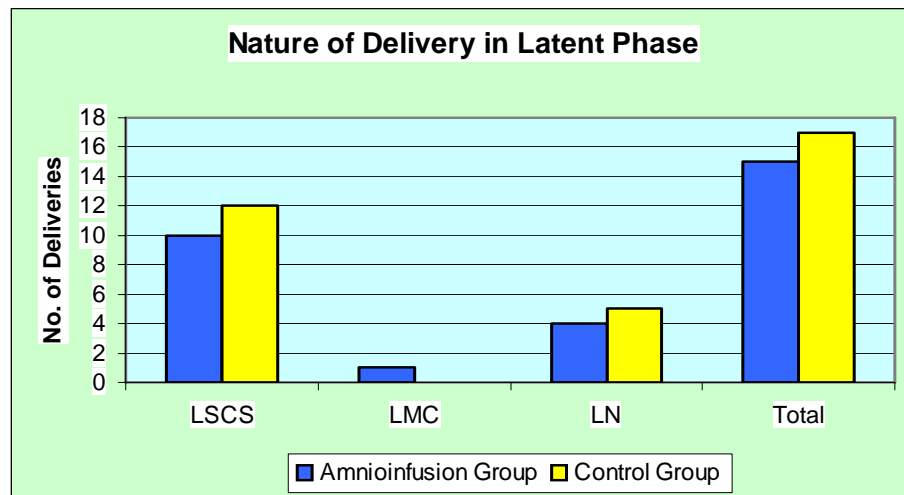
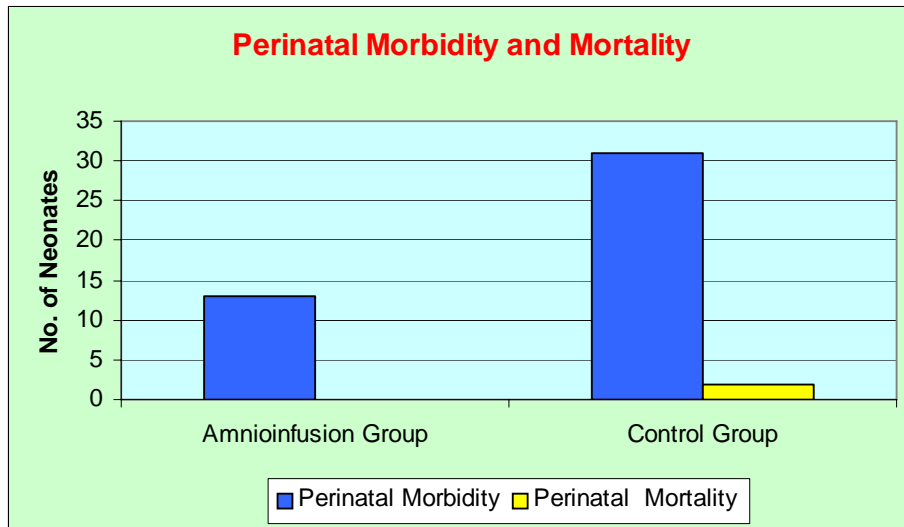




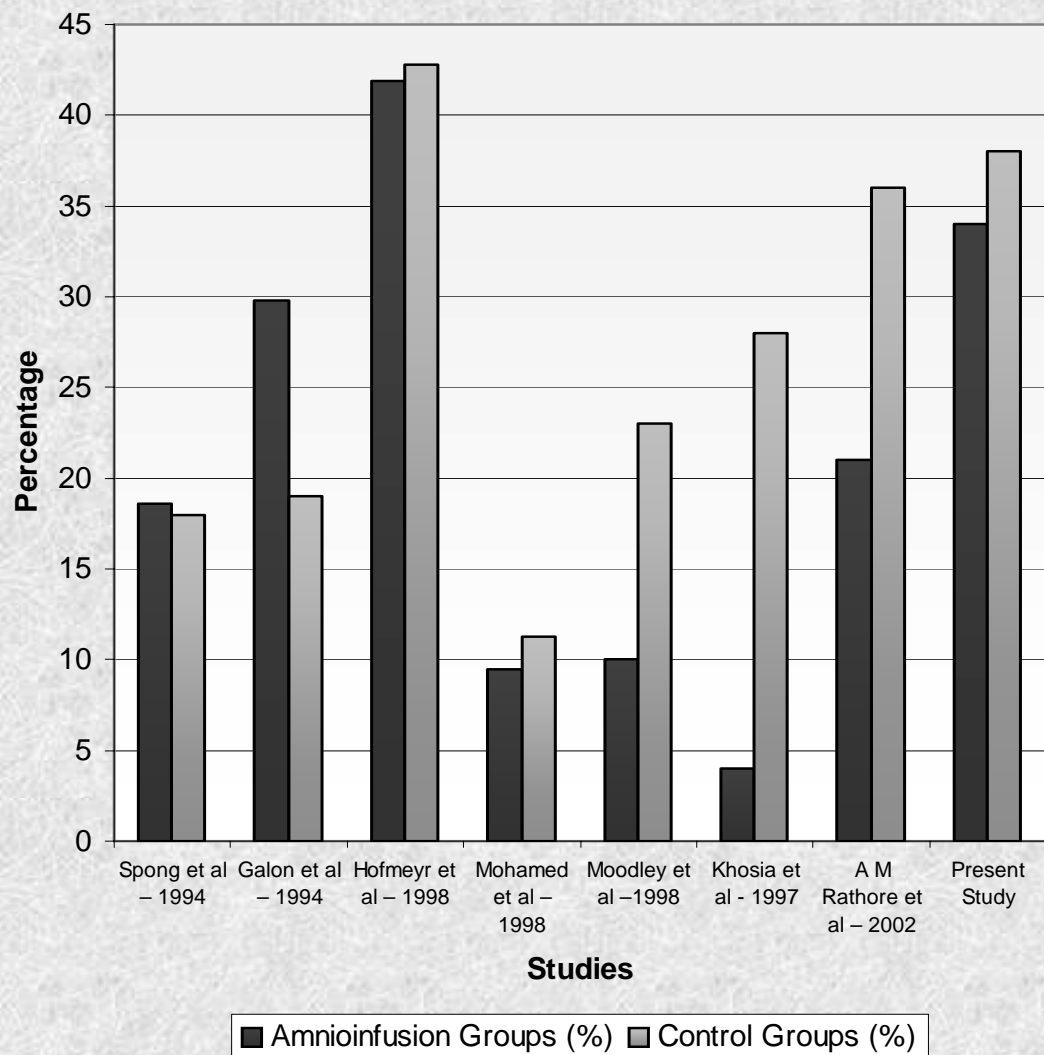


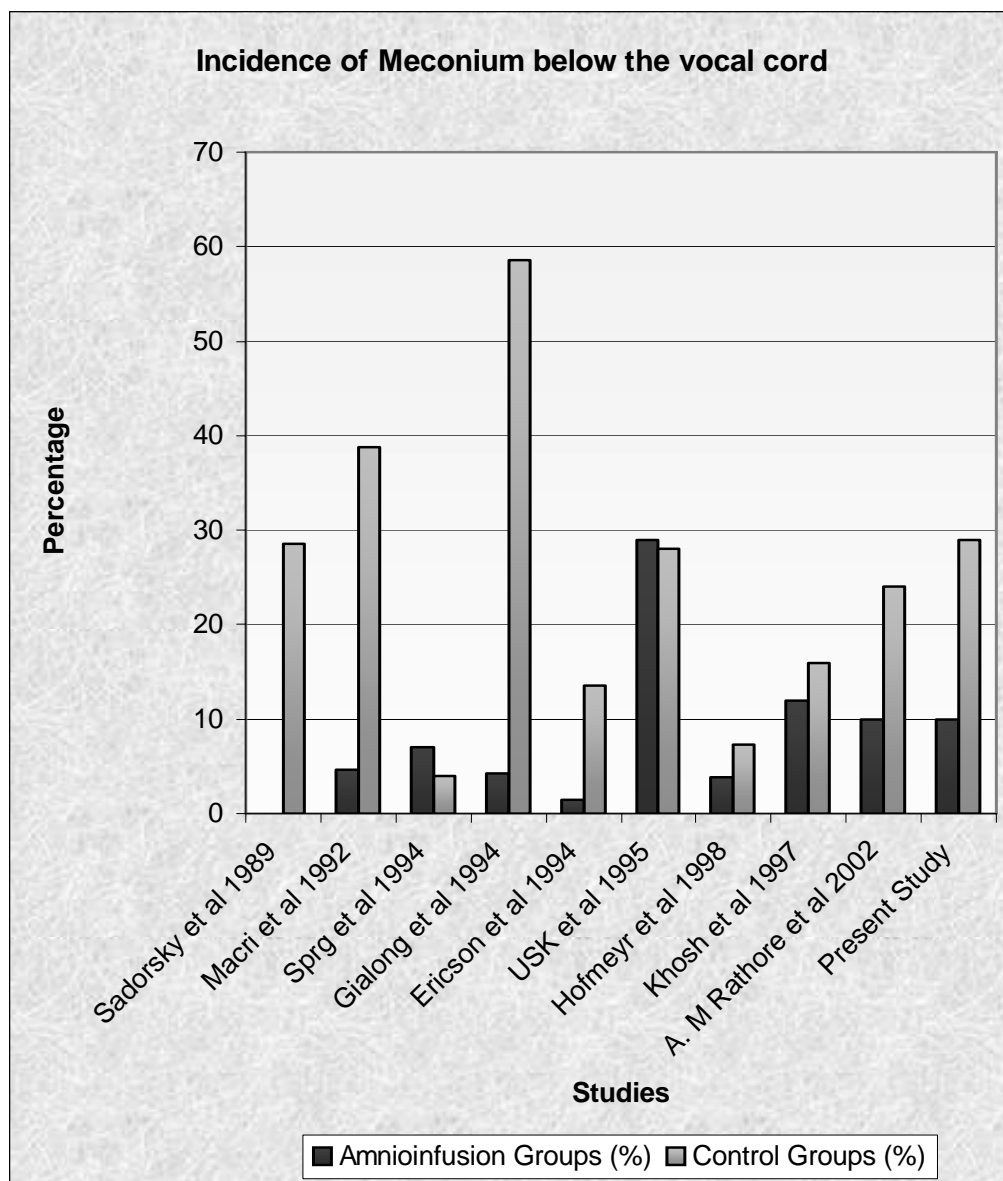






### Intrapartum Amnioinfusion and LSCS rates





### Incidence of Meconium Aspiration Syndrome

